SCIENTIFIC SYMPOSIA ON ETS IN THE 1980S

A number of international conferences and scientific symposia have addressed the issue of ETS in the 1980s. These conferences featured presentations and discussions from scientists, physicians and environmental researchers from around the world. Contrary to the conclusions of the EPA Review Draft on ETS, the conclusion common to many of the various conference organizers is that the claim that ETS causes disease in nonsmokers is not scientifically justified. For example:

5. In 1983, the University of Geneva hosted an international workshop to discuss scientific developments relating to ETS. The organizer of the symposium summarized their discussions as follows:

> An overall evaluation based upon available scientific data leads to the conclusion that an increased risk [of lung cancer] for nonsmokers from ETS exposure has not been established.[1]

6. In May of 1983, scientists and epidemiologists who attended a workshop on "Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies" sponsored by the U.S. National Institutes of Health concluded:

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> A review of the data from the studies which have been carried out or are in progress which address the effect of passive smoking on the respiratory system suggests that the effect varies from negligible to quite small.[2]

7. Similarly, organizers of a symposium on the "Medical Perspectives on Passive Smoking" held in Vienna in April, 1984 and co-sponsored by the German and Austrian societies for industrial medicine in cooperation with the International Green Cross, concluded in a press release:

> Should lawmakers wish to take legislative measures with regard to environmental tobacco smoke, they will, for the present, not be able to base their efforts on a demonstrated health hazard from environmental tobacco smoke.[3]

8. In the preface to the 1986 reprint of those proceedings published by the International Green Cross, Dr. Joseph Handler, a former Director of the World Health Organization (WHO), observed that "the question as to whether the conceivable theoretical possibility of a risk [from ETS] calls for preventive

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measures is not a medical but rather a politico-social one."[4]

9. In October, 1986, an international gathering of scientists in Essen, Germany, considered the experimental and toxicological findings regarding ETS.[5] Professor G.J. Gostomzyk, director of the Health Bureau in Augsburg, Germany, concluded in his review of the proceedings that "so far, even toxicology has not been able to ascertain with any greater degree of probability than did epidemiology that there exists a link between damage to health and passive smoking."[6]

10. In May of 1988, organizers of a symposium on ETS in Austria entitled "Illness Due to Passive Smoking?" issued a press release in which they concluded that "a causal relationship between ETS and illness cannot be established" and that "there is no positive evidence that cancer and other such illnesses are caused by passive smoking."[7]

11. In his introduction to the proceedings of a symposium on indoor air quality held in Bariloche, Argentina in December 1988, Dr. Osvaldo Fustinoni, vice-

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president of the National Academy of Sciences in Buenos Aires, wrote that because of the uncertainty regarding scientific data on ETS exposures and health effects, "it is in question to what extent governments should establish regulations affecting individual freedom, legislating over private facilities like factories or cinemas, theatres, etc."[8]

12. In his "Summary and Concluding Remarks" from the Proceedings of the International Symposium on ETS at McGill University 1989, (Montreal, Canada), co-organizer Dr. Joseph Wu concluded that the published data on ETS, when critically examined and evaluated, do not provide a scientific justification for the claim that ETS is a cause of disease in nonsmokers.[9]

13. In the Proceedings of the International Conference on Indoor Air Quality in Tokyo, Japan, published in 1990, Dr. Hitoshi Kasuga, organizer of the conference, concluded:

> Most participants were of the opinion that it would be very difficult at the present time to reasonably establish a correlation between passive smoking and lung cancer because

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> a detailed examination of all published data (statistical bias in relationship between ETS and lung cancer, estimated ETS levels, incidence of lung cancer in nonsmokers, histological types, etc.) shows that the relationship, if one exists at all, is very slight. Others, however, strongly asserted that there is a correlation.
>
> It was also suggested that there are many other problems in the area of public health, and it would be better to direct our energies towards solving them rather than ETS, as the correlation between it and lung cancer is thought to be slight. The conference, however, felt that steady efforts should be continued to determine the relationship between passive smoking and lung cancer by introducing an objective method of measuring ETS exposure and by thorough research into the cancer site and histological type of nonsmoking cancer patients.[10]

Based upon these conclusions, it is clear that many eminent scientists and physicians do not support the opinions and conclusions of the current EPA Review Draft on ETS.

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2. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, "Report of Workshop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies, May 1-3, 1983," December, 1983.

3. Valentin, H. and E. Wynder, "Health Danger Through Passive Smoking Not Proven: Physicians View on Passive Smoking," (Press release for Symposium on "Passive Smoking from a Medical Point of View," April 9-12, 1984, Vienna, Austria).

4. Handler, J., Preface, Passivrauchen aus medizinischer sicht [Medical Perspectives on Passive Smoking] (Geneva: IRL Imprimeries Reunies Lausanne, 1986): A-B.

5. "Proceedings of the International Experimental Toxicology Symposium on Passive Smoking," ed. E. Mohtashamipur, Toxicology Letters 35(1): 1-168, 1987.

6. Gostomzyk, G., "Passive Smoking-Report on an International Symposium (23-25 October 1986)," Public Health 49: 212-215, 1987.

7. Lehnert, G. and E. Wynder, "Illness Due to Passive Smoking?," (Press Release, Vienna, Austria: May 3, 1988). Translation.

8. Fustinoni, O., "The Problem of Indoor Air Pollution: A Warning to Society," Indoor Air Quality: Symposium (Buenos Aires, National Academy of Sciences of Buenos Aires, 1989): 13-16.

9. Wu, J., "Summary and Concluding Remarks," Environmental Tobacco Smoke: Proceedings of the International Program at McGill University 1989 (Lexington, Mass., DC Health, 1990): 367-375.

10. Kasuga, H., "Outline of the International Conference on Indoor Air Quality," Indoor Air Quality, H. Kasuga (ed.), (Berlin, Springer-Verlag, 1990): 509-510.

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THE DOUBLE STANDARD

In June 1990, the EPA released a Workshop Review Draft entitled "Evaluation of the Potential Carcinogenicity of Electromagnetic Fields." This report concluded:

> The association between cancer occurrence and exposure to either ELF or RF fields is not strong enough to constitute a proven causal relationship, largely because the relative risks in the published reports have seldom exceeded 3.0 in both childhood residential exposures and in occupational situations (6-2).

The Draft evaluated 40 human epidemiological studies (1-2). To reach their overall conclusion, the authors of the Draft carefully assessed the epidemiological data. The Draft concluded:

> . . . because the strength of the association is not strong, a dose-response with magnetic field strength has not been firmly established and potentially confounding influences have not entirely been ruled out, a sufficiently strong case for causality has not yet been made.

The Draft adds:

> However, the fact that the odds ratios are small and in many cases not statistically significant indicates that the association may not be strong and therefore argues against a causal relationship.

In addition, the authors examined animal and short-term tests, arguing that:

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Both animal and in vitro studies are needed to discover the relevant exposure factors and their interaction and to gain some understanding of the mechanisms of action.

On the basis of an overall review of the scientific literature, the authors of the Report concluded that there was inadequate evidence to designate electromagnetic fields as a "probable human carcinogen."

That same month, the EPA released an External Review Draft on the "Health Effects of Passive Smoking." This Review concluded that ETS is a Group A carcinogen, i.e., a known human carcinogen.

The report reviewed 24 human epidemiological studies on ETS and lung cancer. The results were combined and a cumulative increased risk of 1.41 was estimated for lung cancer among nonsmoking wives of smokers compared to nonsmoking wives married to nonsmokers.

No animal inhalation studies or short-term (in vitro) tests were evaluated in the Report. (Published studies on animals report no significant increase in tumors among animals exposed to sidestream smoke; short-term (in vitro) tests on body fluids of nonsmokers exposed to ETS report no significant increases in mutagenic activity compared with nonexposed nonsmokers.)

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Based upon what the authors of this draft characterize as an argument from the overall "weight of the evidence," ETS is deemed a Group A carcinogen.

An obvious "double standard" has been used by the EPA in its evaluation of the scientific literature. In one instance, it is suggested that 40 studies with equivocal evidence from animal and short term tests provide insufficient support for the determination of ELF as a "probable" carcinogen. In the other instance, 24 studies with half the estimated increased risk and no support from animal and short term tests are deemed sufficient to establish ETS as a known human carcinogen. How is the public expected to reconcile this obvious inconsistency?

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ETS STUDIES WHICH WERE NOT CONSIDERED IN THE EPA DRAFT RISK ASSESSMENT

A number of important studies on ETS were not considered in the EPA Review Draft. These studies (1) indicate that nonsmoker exposure to ETS is negligible; (2) demonstrate that epidemiologic studies which suggest an association between ETS exposure and disease are subject to various methodological problems; and (3) provide data which do not support the claim that ETS is associated with disease in nonsmokers.

All of these studies were cited in the EPA's 1989 Indoor Air Reference Bibliography (cover page attached) and were presumably available to, but not considered by, the authors of the Draft Risk Assessment. Omission of those studies has seriously compromised the objectivity of the Review Draft.

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CITATIONS INCLUDED IN INDOOR AIR REFERENCE BIBLIOGRAPHY (EPA/600/8-89/067F, JULY 1989) BUT OMITTED IN REVIEW DRAFT OF HEALTH EFFECTS OF PASSIVE SMOKING (EPA/600/6-90/006A, MAY 1990)

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Washington DC 20460

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APPENDIX I

RECENT PUBLICATIONS ON ETS

The authors of the EPA Review Draft failed to consider a number of pertinent studies, reviews, analyses and criticisms on ETS which were available in the published literature at the time the Review Draft was written. (See comments re: Lung Cancer, Exposure Studies and Children's Studies).

In addition, a number of studies and reviews on ETS have been published recently which would warrant inclusion in any subsequent revision of the Draft. These studies and reviews, together with selected summaries and quotations, are listed below.

I. LUNG CANCER:

A. Primary Studies

1. Kabat, G. "Epidemiologic Studies of the Relationship Between Passive Smoking and Lung Cancer", Proceedings, Toxicology Forum 1990 Annual Winter Meeting, Washington, D.C., February, 1990: 187-199.

Preliminary results from the on-going American Health Foundation study of 90 nonsmoker lung cancer cases indicate no statistically significant associations for lung cancer among males or females who were exposed to ETS during childhood or adulthood (in the home or the workplace).

2. Sobue, T. "Association of Indoor Air Pollution and Passive Smoking with Lung Cancer", Gan no Rinsho 36(3): 329-333, 1990. Translation.

Sobue and colleagues, in a study of lung cancer among non-smoking women in Osaka, Japan, reported no statistically significant associations between ETS exposures during childhood or adulthood and lung cancer among nonsmokers. The researchers noted, however, a statistically significant increased risk of lung cancer among women who had used wood or straw as cooking fuels.

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3. Janerich, D. et al, "Lung Cancer and Exposure to Tobacco Smoke in the Household", NEJM 323(10): 632-636, 1990.

In a highly publicized article, researchers from the U.S. reported that household exposure of 25 or more smoker years during childhood and adolescence could double the risk of lung cancer. However, the study noted that exposures to spousal smoking, smoking in the workplace, and smoking in social settings were not significantly associated with nonsmoker lung cancer incidence, nor were cumulative exposures during adulthood or over a lifetime.

B. Studies on Confounding Factors

1. Rylander, R., et al., "Lung Cancer Risk by Oral Exposure," Present and Future of Indoor Air Quality, C.J. Bieva, et al. (eds.), (Amsterdam: Excerpta Medica, 1989): 91-100.

"A risk factor of 1.3 was found for black tea and of 2.4 for green tea drinkers regardless of smoking habits."

2. Tewes, F.J., "Lung Cancer Risk and Mutagenicity of Tea," Environmental Research (52): 23-33, 1990.

"Analysis of the data demonstrated an adjusted and statistically significant increased lung cancer risk of 2.7 among those who drank green tea. Several brands of tea commonly drank in Hong Kong were assayed for mutagenicity using Ames assay. Significantly elevated levels of mutagenic activity were found to be present after metabolic activation using cell-free extracts of cecal bacteria from rats. These data suggest that further research is needed to understand the possible consequences to human health of ingestants taken at low doses but long duration over the normal lifespan,

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C. Published Critiques and Issue Analyses

1. Layard, M., "Assessing the Validity of a Japanese Cohort Study," Present and Future of Indoor Air Quality, C.J. Bieva, et al. (eds.), (Amsterdam: Excerpta Medica, 1989): 177-180.

> "Our analyses strongly suggest that reported death rates in the Hirayama cohort were substantially lower than Japanese national rates because mortality among persons lost to follow-up was higher than among those successfully traced."

2. Kilpatrick, S., "An Example of Extra-Poisson Variation Suggesting an Under-Specified Model," Present and Future of Indoor Air Quality, C.J. Bieva, et al. (eds.), (Amsterdam: Excerpta Medica, 1989): 83-90.

3. Wynder, E. and Kabat, G., "Environmental Tobacco Smoke and Lung Cancer: A Critical Assessment," Indoor Air Quality, H. Kasuga, (ed.), (Berlin-Heidelberg: Springer-Verlag, 1990): 5-15.

> "No consistent association has been reported for lung cancer and exposure to ETS in childhood, which might be expected to exert a greater effect, especially when followed by exposure throughout adulthood. Of course recall of ETS exposure in childhood is more difficult than recall of such exposure in adulthood."

4. Katzenstein, A., "An Estimate of Adult Mortality in the United States from Passive Smoking; A Response," Environment International, 16: 175-193, 1990.

5. Mantel, N., "What Is the Epidemiologic Evidence for a Passive Smoking-Lung Cancer Association?" Indoor Air Quality, H. Kasuga, (ed.), (Berlin-Heidelberg: Springer-Verlag, 1990): 341-347.

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6. Rylander, R., "Environmental Tobacco Smoke and Lung Cancer," The New England Journal of Medicine 323 (12): 834, 1990.

"Studies evaluating the hypothesis of a relation between exposure to environmental tobacco smoke and lung cancer must take into account other environmental risk or protection factors and the possibility that exposure to environmental tobacco smoke may be confounded. This has not been considered in the majority of such studies. Until this has been done, the claim of causality between environmental tobacco smoke and lung cancer remains uncertain."

7. Faccini, J., "The Role of Histopathology in the Evaluation of Risk of Lung Cancer from Environmental Tobacco Smoke," Exp. Fathol 37 (1-4): 177-180, 1989.

"Current clinical practice for treating lung cancer does not provide adequate histopathological evaluation for the clear distinction between primary and metastatic neoplasia. Reliance on such clinical diagnosis jeopardizes the scientific validity of many epidemiological studies designed to assess the risk of inhalation of environmental tobacco smoke (ETS)."

8. Layard, M., "Environmental Tobacco Smoke and Cancer: The Epidemiologic Evidence," Environmental Tobacco Smoke, Proceedings of the International Symposium at McGill University 1989, J. Ecobichon, (ed.), (Lexington, Mass.: D.C. Health, 1990): 99-115.

9. Mantel, N., "The Passive Smoking Myth," Present and Future of Indoor Air Quality, C.J. Bieva, et al. (eds.), (Amsterdam: Excerpta Medica, 1989): 155-159.

10. Lee, P., "Passive Smoking and Lung Cancer; Fact or Fiction?" Present and Future of Indoor Air Quality, C.J. Bieva, et al. (eds.), (Amsterdam: Excerpta Medica, 1989): 119-128.

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11. Adlkofer, F., "Exposure to ETS and its Biological Effects: A Review," Present and Future of Indoor Air Quality, C.J. Bieva, et al. (eds.), (Amsterdam: Excerpta Medica, 1989): 189-196.

12. Uberla, K. and Ahlborn, W., "Passive Smoking and Lung Cancer: A Reanalysis of Hirayama's Data," Indoor Air Quality, H. Kasuga (ed.), (Berlin-Heidelberg: Springer-Verlag, 1990): 333-340.

13. Letzel, H. and Uberla, K., "Meta-Analyses on Passive Smoking and Lung Cancer," Indoor Air Quality, H. Kasuga (ed.), (Berlin-Heidelberg: Springer-Verlag, 1990): 316-322.

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1. Watts, R., et al., "Cotinine Analytical Workshop Report: Consideration of Analytical Methods for Determining Cotinine in Human Body Fluids as a Measure of Passive Exposure to Tobacco Smoke," Environmental Health Perspectives, 84: 173-182, 1990.

2. Vaughan, W., "Impact of a "Designated Smoking Area" Policy on Nicotine Vapor and Particle Concentrations in a Modern Office Building," J. Air Waste Manage Assoc., 40, (7): 1012-1017, 1990.

3. Haley, N., "Elimination of Cotinine from Body Fluids: Disposition in Smokers and Nonsmokers," AJPH 79, (8): 1046-1048, 1989.

> "Differences in the mode of uptake and absorption of nicotine and possible differences in nicotine metabolism may play roles in the clearance rate differences between smokers and nonsmokers."

4. Von Meyerinck, L., et al., "Exposure of Rats and Hamsters to Sidestream Smoke from Cigarettes in a Subchronic Inhalation Study," Exp. Pathol, 37 (1-4): 186-189, 1989. Poster.

> "A 90-day feasibility study was performed in which rats and hamsters were exposed to the sidestream smoke of cigarettes. The only histopathological changes observed were hyperplasia and metaplasia of the epithelium covering the dorsal nasal turbinate bones in rats. These effects were reversible within 90 days."

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5. Scherer, G., "Biomonitoring of Exposure to Potentially Genotoxic Substances from Environmental Tobacco Smoke," Environment International, 15: 49-56, 1989.

"No statistically significant increase was found in the urinary mutagenicity after either of the two exposure regimens, whereas thioether excretion was significantly elevated. The data suggest that nonsmokers in real-life situations take up very low doses of ETS constituents, and detoxification of the genotoxic substances inhaled is effective."

6. Leaderer, B., "Assessing Exposures to Environmental Tobacco Smoke," Risk Analysis, 10, (1): 19-26, 1990.

7. Oldaker, G., et al., "Results from Surveys of Environmental Tobacco Smoke in Offices and Restaurants," Indoor Air Quality, H. Kasuga (ed.) (Berlin-Heidelberg: Springer-Verlag, 1990): 99-104.

8. Sterling, T.D., "ETS in Offices and When Smoking Is Restricted to Designated But Not Separately Ventilated Areas," Indoor Air Quality, H. Kasuga (ed.) (Berlin-Heidelberg: Springer-Verlag, 1990): 120-129.

9. Baker, R., "The Origins and Properties of Environmental Tobacco Smoke," Environment International, 16: 231-245, 1990.

10. Coultas, D., "Questionnaire Assessment of Lifetime and Recent Exposure to Environmental Tobacco Smoke," American Journal of Epidemiology, 130, (2): pp. 338-347, 1989.

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11. Coultas, D. et al., "A Personal Monitoring Study to Assess Workplace Exposure to Environmental Tobacco Smoke," AJPH, 80, (8): pp. 988-990, 1990.

"The controversial effects of involuntary smoking in the workplace need further investigation. The conduct of such research would be facilitated by the development of unintrusive and accurate methods of exposure assessment. Alternative approaches include active and passive monitoring, biological markers, and questionnaires. We have shown that personal monitoring for tobacco smoke components can be accomplished in the workplace."

12. Perry, R. et al., "Indoor Air Quality: The Contribution of Environmental Tobacco Smoke," Indoor Air Quality, H. Kasuga (ed.) (Berlin-Heidelberg: Springer-Verlag, 1990): 62-75.

13. Piade, J. et al., "Assessment of ETS Impact on Office Air Quality," Indoor Air Quality, H. Kasuga (ed.) (Berlin-Heidelberg: Springer-Verlag, 1990): 112-119.

14. Robertson, G., "Source, Nature, and Symptomology of Indoor Air Pollutants," Indoor Air Quality, H. Kasuga (ed.) (Berlin-Heidelberg: Springer-Verlag, 1990): 393-402.

15. Quackenboss, James J., "Indoor-Outdoor Relationships for Particulate Matter: Exposure Classifications and Health Effects," Environment International, 15: 353-360, 1989.

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APPENDIX II

CONFOUNDERS

The effect of positive confounding must be taken into account when evaluating a possible association in an epidemiological study. The EPA Review Draft does not adequately address the issue of confounding variables in its discussions of epidemiologic studies on lung cancer incidence or children's respiratory disorders. Correlations of confounders with ETS exposure could give rise to spurious associations between ETS exposure and lung cancer incidence or the occurrence of respiratory conditions. Therefore, the effect of each confounder must be considered before a conclusion about the causal role of any one can be reached.

Potential confounding factors are numerous and varied. It is difficult to control for even a few of them in the design of an epidemiological study. The following pages present brief descriptions from the published literature of some of the potential confounders associated with lung cancer and with children's respiratory disorders, followed by lists of references.

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Section A: Lung Cancer

1. Household heating and cooking sources.

The use of rapeseed oil for cooking (as opposed to soybean oil) has been associated with a statistically significant increased risk of lung cancer in Shanghai Chinese women (Gao, et al., 1987). The cooking techniques of stir frying, deep frying and boiling were also associated with increased lung cancer risk in this study.

Several papers have reported on a potential relationship between increased lung cancer risk and the use of kerosene, gas, coal or liquefied petroleum gas heating and cooking sources in Oriental women (Anonymous, 1986; Chapman, et al., 1988; deSerres and Matsushima, 1986; Du, 1990; Mumford, et al., 1987). The Chapman, et al., and Mumford, et al., papers report on a large epidemiologic survey of the population of Xuan Wei, China. The authors found that female lung cancer mortality was strongly associated with the use of "smoky" coal in the home.

A recent paper (Sobue, et al., 1990) reported a statistically significant increased risk of lung cancer among Japanese women who had used straw or wood as cooking fuels.

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2. Diet.

In several papers on nonsmoking Hong Kong Chinese women married to smokers, Koo (1988, 1989 and Koo, et al., 1988) reported increased lung cancer risk among those women who consumed smoked, salted, cured, pickled, processed, or spicy foods. A reduction in risk was reported in those women consuming fresh foods. Additionally, wives with smoking husbands consumed more processed and spicy foods and ate fewer fresh fruits and vegetables than did those whose husbands did not smoke. Maintenance of a traditional diet may explain the observation that Chinese women have high rates of lung cancer regardless of their place of residence (Hong Kong, Shanghai, Hawaii) (Koo, 1989).

In a 1989 paper, Sidney, et al., reported that nonsmokers married to smokers had lower intakes of dietary carotene. The authors concluded that dietary carotene was an important confounder which should be measured in all studies of ETS exposure and lung cancer incidence.

Rylander, et al., (1989) and Tewes, et al., (1990) reported elevated risks associated with the consumption of green or black tea.

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3. Health and medical history.

A study of Shanghai Chinese women by Gao, et al., (1987) reported an association between short (less than 30 days) menstrual cycle length and increased risk of lung cancer. Previous history of respiratory disease (including tuberculosis, pneumonia or emphysema) has been associated with increased risk for lung cancer (Gao, et al., 1987; Wu, et al., 1985).

Family history of lung cancer has also been associated with increased lung cancer risk (Wu, et al., 1988).

At least one hobby, keeping pet birds, has been associated with increased lung cancer risk in a study from the Netherlands (Holst, 1988).

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Section B: Children's Respiratory Disorders

1. Household heating and cooking sources.

Children living in households with gas stoves have been reported to have a greater history of respiratory illness before the age of two and small but significantly lower levels of FEV_1 and FVC corrected for height (Speizer, 1980). Similarly, exposure of children to gas cooking in the first two years of life has been associated with an increased risk of hospitalization for respiratory illness (Ekwo, et al., 1983). Lebowitz (1984) reported associations of gas stove use with daily peak flow in asthmatic, normal, and allergic subjects.

Oxides of nitrogen arising from the use of gas stoves for cooking were proposed to be related to a reported increase in cough, "colds going to the chest," and bronchitis in a study of 5,758 English and Scottish children aged six to eleven years (Melia, et al., 1977). A number of other confounders were controlled for in this study, including "age, social class, latitude, population density, family size, overcrowding, outdoor levels of smoke and sulphur dioxide and types of fuel used for heating." Melia (1979) reported similar results for a five-year longitudinal study of 4827 boys and girls, ages five to ten years. This reported association, although apparently independent of age, sex, social class, number of cigarette smokers in the home, and latitude, was only found in urban areas.

Use of unvented kerosene heaters, which release NO_2 into the indoor environment, was associated with significantly more days of acute respiratory illness in exposed children (Berglund, et al., 1984). In this study, there was no difference in the number of cigarettes smoked daily in the homes of exposed versus unexposed children. NO_2 exposure was also reported to be associated with a risk of reporting lower respiratory symptoms in children under the age of seven (Berwick, et al., 1989).

Osborne and Honicky (1989) reported increased proportions of chest illnesses and hospitalizations for chest illness before age two in young children living in homes heated by wood-burning stoves. Medical histories, sociodemographic factors, or exposure to other pollutant sources did not account for the reported association.

Hot water heating systems were reported to have a large effect on lung function in children, when compared to forced air heating and air conditioning systems (Hosein, 1987).

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2. Outdoor air pollution.

Outdoor air pollutants have been described as confounders in certain studies. For instance, acute respiratory disease incidence was reported to be positively associated with higher ambient sulfate levels (Harrington and Krupnick, 1985).

Kerigan, et al., (1986) examined the importance of indoor and outdoor environmental factors (parental smoking, gas cooking, suspended particulates and sulfur dioxide) in the respiratory health of seven- to ten-year-old Canadian children. The researchers were unable to distinguish between the effects of parental smoking and gas cooking because the prevalence of these variables was highest in an industrial area of high particulate pollution.

Pope (1989) reported a strong association between respiratory illness and particulate pollution in children living in a study site which experiences relatively high levels of particulate pollution.

A study comparing Israeli children living in a polluted industrial town versus those living in an unpolluted area reported that chronic respiratory symptoms and most pulmonary diseases were significantly more common among those children from the polluted town (Goren and Hellmann, 1988).

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3. Organic substances.

The relevance of home dampness in the etiology of respiratory symptoms in children is supported by current research which links dampness with the presence of molds, dust mites, fungi and other allergenic microbes.

Odds ratios of 1.23 and 2.16 were reported for home dampness after adjustment for several factors, including maternal smoking, in a cohort study of 4,625 eight- to twelve-year-old children living in six United States cities (Brunekreef, et al., 1989). The authors reported odds ratios for molds of 1.27 to 2.12 after adjustment for maternal smoking and several other factors.

Another study (Martin, et al., 1987) reported higher rates of respiratory symptoms and symptoms of infection and stress among children living in damp houses. The presence of "fungal mould" was also reported to be related to higher rates of respiratory symptoms, independent of smoking in the household. In another study, the growth of fungi and molds in the home was directly related to respiratory symptoms and sensitization to common allergens in children (Ownby and McCullough, 1988).

Platt, et al., (1989) reported that children living in damp and moldy dwellings had a greater prevalence of respiratory symptoms and headache and fever than those living in dry homes. The authors reported a dose-response relationship with increasing numbers of symptoms reported in dwellings with higher severity of dampness and mold. All these differences persisted after controlling for possible confounding factors such as household income, cigarette smoking, unemployment, and overcrowding.

Atopic sensitization of children to house dust mites was reported to be related to home dampness (Nordvall, et al., 1988).

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4. Demographic, medical and socioeconomic factors.

Low socioeconomic status has been associated with an increased incidence of respiratory complications (Colley, 1975; Kerigan, 1986). Factors related to lower socioeconomic status include: inadequate medical care, poor nutrition, parental neglect, poor outdoor air quality, increased parental coughing, higher gas stove usage, frequent change of address, and lower per capita living space. In a study of 1,050 European children aged eight and nine years, lifetime and current prevalence of wheeze were both significantly higher in children from low socioeconomic status (Mitchell, et al., 1989).

Watkins, et al., (1986) reported high consultation rates for respiratory illness in children whose fathers were in manual occupations. This association was not explained by crowded home conditions or parental smoking. Gardner, et al., (1984) reported significantly higher rates of lower respiratory disease in infants of low socioeconomic status.

Cross-infection also plays a role in the incidence of children's respiratory disease. For instance, in a 1988 paper, Koo, et al., reported that among Japanese and Hong Kong Chinese women, there was a highly significant correlation between the frequency of maternal respiratory illness and the frequency of respiratory illness in her children.

Cross-infection may be relevant to the reports of associations between day care attendance and respiratory illness. Anderson, et al., (1988) reported that care outside the home (day care) is an important factor for acquiring lower respiratory tract illness and infectious diseases in children under two years of age. Gardner, et al., (1984) also noted significantly higher rates of lower respiratory disease among day care infants. Fleming, et al., (1987) reported an increased risk for upper respiratory tract infection associated with day care attendance.

Familial characteristics and genetics may also act as confounders. For instance, in a 1982 publication, Lebowitz, et al., report that an observed relationship between children's pulmonary function and parental smoking disappeared when household aggregation of body mass was taken into account. Another Lebowitz, et al., study (1984) also reported that there was "no remaining independent aggregation of pulmonary function measurements" after familial aggregation of body habitus was controlled for. Genetic predisposition may play a role in respiratory illness and pulmonary function (Colley, et al., 1974); although cross-infection is also involved (Colley, 1974).

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"Lifestyle" may also act as a confounder. A study in Copenhagen (Holma and Winding, 1977) examined 109 social, medical, housing, and hygiene factors on morbidity. The best predictors for health were "thriving" (satisfaction), followed by "housing standard" and "personal hygiene." The authors found no effect of parental cigarette smoking on the morbidity of young children. A survey of 314 nonsmoking Hong Kong Chinese women and their children and 243 Japanese women and their children reported that chronic cough and sputum symptoms were at least 10 times more prevalent in Hong Kong (Koo, 1988). This observation was attributed to occupational exposure to dust or fumes and larger household sizes among the Hong Kong mothers.

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